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ATTENTION DEFICIT HYPERACTIVITY DISORDER

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PSYCHOLOGY TEACHERS UPDATE

Psychology Teachers Update is designed to give a brief overview of the main developments in the different areas of psychology. There is a proliferation of journals and research, and it is very difficult to keep abreast of the latest trends, particularly in the many and varied areas of psychology.

Each issue of Psychology Teachers Update will cover particular topicS, and summarise the main research directions and findings in the last ten to fifteen years approximately. The aim is to give teachers the feel of what is happening in that area of psychology.

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A complete list is available at http://kmbpsychology.jottit.com

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INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is one of the more controversial conditions with academic opinions varying from a cultural construction to the search for the biological basis. When amphetamine-based stimulant drugs are prescribed for it, the debate becomes even more heated.

The increasing rate of diagnosis of ADHD and increased stimulant use is behind the public debate over the issues like the existence of the condition (ie: the validity of diagnosis), the causes, and the ethics of stimulant medication use with children (Singh 2008).

A number of positions are taken in the debate (Singh 2008):

- ADHD is a valid category of mental disorder with a biological basis (and thus medication is the appropriate treatment);
- ADHD is caused by biological and social factors together, and the diagnostic category is not completely valid. Medication can be used, but so can therapies, like behaviour modification;
- ADHD is a valid diagnosis, but the causes are environmental (eg: food additives, maternal smoking). Thus prevention is more important than medication;
- ADHD is not a real disorder.

In 2002 a consensus statement on ADHD was published by professionals and researchers involved with it (Barkley et al 2002). "However, the statement could probably best be described as a position statement because the diverse views of what ADHD is and what should be done about it were not reflected or represented" (Cannon et al 2004).

In a special debate in the "British Journal of Psychiatry" in 2004, both sides of the argument were presented. Sami Timimi (2004) argued that there are "no specific cognitive, metabolic or neurological markers and no medical tests for ADHD". ADHD as a medical condition is unhelpful and stifles diversity, particularly when prescribing stimulants for them.

In opposition, Eric Taylor (2004) pointed out that "individual differences in hyperactivity have known physical counterparts; in brain structure and function and DNA composition", though he accepted the role of social factors in the degree of hyperactivity shown by

children.

This is a hotly debated topic, and, sadly, Taylor admitted he had "received menaces from an anti-psychiatry organisation". The heat of the debate does push both sides further apart. The "science or "pro" side emphasise that ADHD is a real disorder, and "treats ADHD diagnosis as though it were a concrete representation of disorder, rather than an abstract approximation" (Singh 2008). All diagnosis of symptoms of mental disorders are approximations to underlying disorders: "there are no laboratory tests to determine unequivocally whether a subject has the disorder" (Singh 2008).

Another issue concerns the identification of ADHD as a risk factor for adult problems. There are concerns about stigmatising individuals with diagnosis as well as the ethics of pre-symptomatic treatments (Singh 2008).

DIAGNOSIS

The core symptoms of ADHD are hyperactivity, inattention, and impulsiveness (table 1). Most diagnosis occurs at school age, and the majority (about three-quarters) of cases are male (Singh 2008).

Children with ADHD can show developmental problems like poor sense of time, impaired planning ability, delayed onset of language, delayed motor co-ordination, and mild deficits in intelligence (Chu 2003b). Note that not all sufferers show developmental problems.

INATTENTION	HYPERACTIVITY	IMPULSIVENESS
• Easily distracted • Attention deficit • Hypoarousal * • Inability to sustain concentration • Leaving tasks unfinished	• Excessive motor activity • Difficulty remaining seated • Noisiness • High level of activity	• Unthinking and impetuous behaviour • Disinhibited • Reckless

(* Reduced response to stimuli that facilitates attention and motivation; Chu 2003a)

Table 1 - Key characteristics of ADHD.

DSM and ICD vary in their diagnostic categorisations:

DSM-IV (APA 1994)

Two primary categories of symptoms (inattention and impulsivity-hyperactivity) [1] and three sub-types (inattentive, hyperactive-impulsive, and combined) [2]. Each type is diagnosed by six or more symptoms from nine (table 2), and onset before age seven years, with symptoms present in at least two settings. ADHD is classified under "Disruptive Behaviour Disorders" along with conduct disorders and oppositional defiant disorder.

ICD-10 (WHO 1992)

This uses the term "Hyperkinetic Disorder" (HKD) not ADHD, and requires all three symptoms of hyperactivity, inattention, and impulsivity together for diagnosis.

Swanson et al (1998) felt that HKD equated to the ADHD combined sub-type.

- Often fails to give close attention to details or makes careless mistakes in school work, work, or other activities (I).
- Often loses things necessary for tasks or activities (eg: school assignments, pencils) (I).
- Often does not seem to listen when spoken to directly (I).
- Is often forgetful in daily activities (I).
- Often talks excessively (H).
- Often blurts out answers before questions have been completed (H).
- Often fidgets with hands or feet or squirms in seat (H).
- Often has difficulty awaiting turn (H).
- Is often "on the go" or often acts as if "driven by a motor" (H).

(I = inattention sub-type; H = impulsivity-hyperactivity sub-type)

(Source: APA 1994)

Table 2 - Examples of behavioural criteria used for diagnosis in DSM-IV.

Diagnosis of ADHD using DSM-IV is 3-4 times more likely than diagnosis of HKD (Singh 2008). Thus prevalence rates can vary throughout geographical regions of the world - from 12% of school-age children (South

[1] It has been suggested that these are separate disorders. Sagvolden et al (2005) described them as Attention Deficit Disorder (ADD) predominantly inattentive type without impulsiveness and hyperactivity, and Hyperactive/Impulsive Disorder with hyperactivity, impulsivity, and problems with sustaining attention which develops into ADHD combined type. The researchers prefer to call the latter "Reinforcement Extinction Disorder" (RED).

[2] Inattentive sub-type = 20-30% of cases, hyperactivity-impulsive sub-type = <15%, and combined = 5-75% (Wilens et al 2002).

American countries) down to 5% (European countries). In the USA, studies vary from 2-18%, and 0.5-26% in the UK (Singh 2008) (table 3).

COUNTRY	PREVALENCE RATES
USA	DSM-IV official rate 5%; 7.5% (most stringent criteria) - 16% (least stringent criteria)
Australia	1.6% inattention sub-type, 0.2% impulsivity-hyperactivity sub-type, 0.6% combined
UK	HKD 0.5-1.5%; ADHD 5%

(Source: Chu 2003a)

Table 3 - General accepted prevalence rates from three countries.

Co-morbidity of ADHD with other conditions is common. In an extensive US study (Jensen et al 2001), only 31% of children had a single diagnosis of ADHD. Co-morbidity with oppositional defiant disorder was diagnosed in 40% of sufferers, tic disorder 11%, conduct disorder 14%, anxiety disorders 34%, and mood disorders 4%.

Table 4 lists the social factors that can influence the variations in prevalence rates of ADHD (Singh 2008).

- Ethnicity
- Educational level
- Social-economic status
- Attitude of practitioner towards ADHD
- Access to psychiatric services in a locality
- Pressure within and on schools
- Parental expectations for child
- Mothering ideology (cultural beliefs about what is good mothering)
- Masculine stereotypes (social expectations about being male, eg: aggression level)
- Government educational policy (eg: additional educational resources for diagnosed children)
- Health service policy (eg: access to treatment after diagnosis)
- Marketing by pharmaceutical companies (eg: direct-to-consumer advertising in USA)
- Social context [3]

Table 4 - Some social factors that lead to the variations in prevalence rates of ADHD.

[3] In interviews with 26 clinicians about diagnosis of ADHD, Rafalovich (2005) found that they did not "practice within a vacuum, but are instead largely affected by the marked scepticism that surrounds ADHD" (p305).

A number of ways of measuring and diagnosing ADHD are used in practice:

- DSM Symptom Checklists - eg: ADHD Rating Scale (Du Paul et al 1998); clinicians rate fourteen symptoms.

- Measures of specific symptoms - eg: Auditory Continuous Performance Test II (Keith 1994); measures sustained auditory attention by requiring the child to listen to a tape for fifteen minutes and respond appropriately to instructions.

- Multi-factor Rating Scales - eg: Child Behaviour Checklist (CBCL) (Achenbach 1991); parents' rating of behaviour problems and social competence generally.

- Observation measures - eg: ADHD Behaviour Coding System (ADHD-BCS) (Barkley 1990); used in clinic playrooms and classrooms.

- Structured interviews - eg: ADHD Parent Interview (Barkley 1990).

HISTORY

"Very hyperactive, restless and inattentive children have been identified by clinicians and medical researchers dating back to at least 1902. Since then, upwards of twenty different diagnostic labels have been used to categorise children who exhibit these problematic behaviours" (Mayes and Rafalovich 2007 p436). Mayes and Rafalovich (2007) emphasised that the symptoms that are seen as problematic have remained similar over the 20th century, even if the label given for them and explanation for the underlying cause varied [4].

Chu (2003a) described the historical developments in the 20th century in four periods:

- 1900-60: from organic deficits to minimal brain dysfunction;
- 1960-70: the development of a symptom-oriented classification system;
- 1970-90: from hyperactive to attention deficit;
- 1990-2000: from attention deficit to dysfunction in self-regulation.

[4] The fact that similar behaviours have been noted in the past is often used to support the idea that ADHD is "real". But individuals in the past were not actually describing ADHD as it did not exist at the time (Singh 2008).

In fact, Alexander Crichton, a Scottish-born physician, described "ADHD-like" symptoms as early as 1798 as "mental restlessness" (Palmer and Finger 2001).

An English paediatrician, Sir George Frederick Still, described the symptoms in 1902 - the children "exhibited violent outbursts, wanton mischievousness, destructiveness and a lack of responsiveness to punishment" with a "quite abnormal incapacity for sustained attention, causing school failure even in the absence of intellectual retardation". Furthermore, "this pattern occurred more often in boys than in girls, became frequently apparent by early school years, was sometimes accompanied by peculiarities of physical appearance, generally showed little relationship to the child's training and home environment, and commonly shared a poor prognosis". Overall, Still saw the problem as a "defect of moral control", but due to "the manifestation of some morbid physical condition" (Still 1902 quoted in Mayes and Rafalovich 2007). This was the first use of this idea as opposed to the traditional view of the time of a "character flaw".

The naming of the physical cause as due to mild brain damage, probably during birth, came from Alfred Tredgold involved in the Royal Commission on Mental Deficiency, and the Mental Deficiency Act 1913. This act enshrined the category of "feeblemindedness" in which "ADHD" children were placed. Tredgold (1922) linked the brain damage to encephalitis lethargica ("sleepy sickness") which came with the post-World War I flu pandemic.

By the 1930s, this explanation was replaced by observations that emphasised the hyperactivity aspect of the problem behaviour (eg: Kahn and Cohen 1934). In time, this idea evolved into the first specific name (in the USA) - hyperkinetic impulse disorder (Laufer et al 1957). These authors explained the disorder through brain damage or injury, specifically to the diencephalon [5].

Subsequently it became clear that most children diagnosed with hyperkinetic impulse disorder did not have such or any brain damage (eg: Chess 1960: fourteen of 82 disordered children had damage). So the idea of minimal brain damage became minimal brain dysfunction (eg: Clements and Peters 1962).

In DSM-II (APA 1968) hyperkinetic impulse disorder was called "hyperkinetic reaction of childhood" as all childhood disorders were called "reactions" in this document. The 1960s also saw the use of stimulants to treat it, especially in the USA (eg: 150 000-200 000

[5] Part of forebrain including, for example, the thalamus and hypothalamus.

children by 1969; Mayes and Rafalovich 2007), and the subsequent controversies over the condition and treatment. For example, in the USA, "The Myth of the Hyperactive Child and Other Means of Child Control" (Schrag and Divoky 1975) argued that "Ritalin" was being used as a "chemical straitjacket" to control children's natural exuberance.

On the other side of the debate, Benjamin Feingold (1975) linked hyperactivity to food additives in "Why Your Child is Hyperactive".

Hyperkinetic became hyperactive in the 1970s. DSM-III (APA 1980) introduced the term "Attention Deficit Disorder" (ADD) with or without hyperactivity, ADHD was added in DSM-IIIR (APA 1987). These terms were combined in the three sub-types of ADHD in DSM-IV (APA 1994).

Recently, ADHD has been presented as a problem of self-regulation and executive functions (ie: more than just inattention and impulsivity) (Barkley 1998).

CAUSES OF ADHD

Recent research has moved away from the hunt for single causes to "identifying complex developmental pathways that link genetic, biological and environmental risk factors to phenotypic expressions in multiple different combinations" (Singh 2008).

GENETICS

Twin studies have been used to establish heritability for ADHD ranging from 60-90%, and the remainder (10-40%) due to non-shared environmental factors (Waldman and Gizer 2006).

In that case, the focus has moved to which genes are inherited. This is studied in two main ways. Association studies compare specific areas of the genome for candidate genes in affected and non-affected individuals. For a candidate gene, there is a choice of possibilities (alleles) (ie: from biological mother or from biological father). One version may be high-risk for the disorder and the other low-risk. It is assumed that the affected group will show more cases of the high-risk allele.

Linkage studies look for certain genes within a biological family, particularly between those members with the disorder and those without.

Different studies tend to find different associations. For example, Brookes et al (2006) found variations in genes related to dopamine transporters and receptors and ADHD. This finding has been replicated, but

the overall effect is small (Singh 2008).

Other findings tend not to be replicated.

A promising candidate gene is known as DAT1 and it codes for a protein involved in dopamine reuptake. As well as human studies, genetically engineered "knockout" mice lacking both copies of DAT1 show "behaviours analogous to ADHD" (Waldman and Gizer 2006).

Another gene related to dopamine, but to the receptors, which has produced interest is DRD4. There are many other candidate genes being studied, particularly related to neurochemistry, but in each case "there is a mixed picture of positive and negative findings" (Waldman and Gizer 2006).

Table 5 summarises the evidence for gene related to dopamine, dopamine and ADHD (Waldman and Gizer 2006).

- Methylphenidate (stimulant medication) used to treat ADHD works by making more dopamine available at the synapse.
- Genetically engineered mice.
- Single photon emission computed tomography (SPECT) is able to show dopamine activity in the brain.
- Association studies: affected vs unaffected children.
- Family studies; eg: affected vs unaffected siblings.

Table 5 - Sources of evidence for the role of dopamine-related genes and dopamine in ADHD.

But a difference in a single gene is not sufficient to cause the disorder. It is more likely that there are "a multitude of susceptibility genes, each contributing only a small magnitude of the overall risk for the disorder" (Waldman and Gizer 2006).

Rather than specific genes being directly involving in causing ADHD, research has focused on endophenotypes. These are intermediates between the gene and the behaviour (figure 1). So the gene caused a certain brain development, say, which causes the symptoms seen.

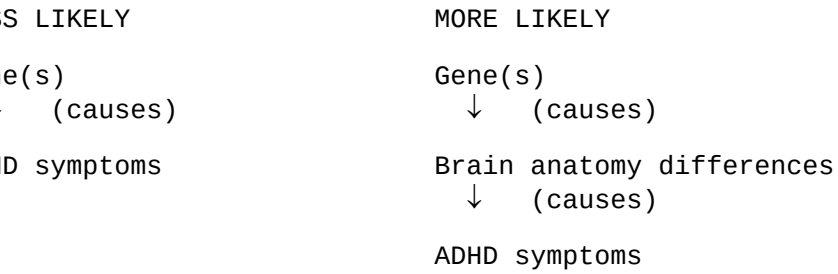

Figure 1 - Possible relationships between genes and ADHD symptoms.

NEUROCHEMISTRY

Sagvolden et al (2005) explained the symptoms of ADHD as due to altered dopamine functioning in different parts of the brain:

- Dopamine underactivity in areas of the limbic system (mesolimbic pathway) produces hyperactivity, impulsiveness, lack of sustained attention, and disinhibition.
- Dopamine underactivity in cortical areas (mesocortical pathway) produces attention problems, and poor executive functions.
- Dopamine underactivity in other areas (nigrostriatal pathway) leads to motor function problems like clumsiness.

The dopamine underactivity can be due to genetics, but also drug abuse and environmental pollutants (Sagvolden et al 2005).

NEUROANATOMY/NEUROPHYSIOLOGY

The development of medical technology in the last quarter of a century has produced sophisticated neuroimaging techniques, like PET scans, which can show the structure and function of the living brains of individuals with ADHD. The structure relates to physical differences in size, say, and function is how the brain works (eg: blood flow patterns).

Differences in structure include, for example, smaller areas in the dorsolateral prefrontal cortex, the corpus callosum, or the cerebellum than controls (Singh 2008). Functional differences have been seen in the frontal cortex (Singh 2008).

But most imaging studies are small-scale (eg: less than twenty participants), and exact findings are not necessarily replicated (Singh 2008). It is also difficult for ADHD sufferers to remain still in scanners for long periods.

Differences in electrical brain activity has also been measured; eg: reduced beta activity in temporal lobes (Chu 2003a).

Voeller (2001) described a control loop within the brain that includes the frontal lobe, basal ganglia and thalamus, and which activates or inactivates other parts of the brain. Children with ADHD have differences in this system. For example, they seek out movement stimulation (ie: hyperactivity) to activate pathways within this

loop. Put another way, ADHD children have underaroused brains, and are hyperactive to rectify it [6].

ENVIRONMENTAL

A number of environmental factors have been proposed as the cause (table 6), but few have been replicated as the sole cause of ADHD. However, they may be triggers of a predisposition for ADHD.

- Family discord
- Lead
- Very low birthweight
- Prenatal environment (eg: mothers takes steroids during pregnancy)
- Diet (eg: food additives)
- Excessive television watching at young age

Table 6 - Examples of environmental factors proposed as causes of ADHD.

The child's environment is crucial in how the problem behaviours in ADHD develop. For example, Patterson (2002) outlined coercive family interactions that lead to problem behaviours generally through the principles of reinforcement. For instance, a child demands immediate attention by shouting which is given by the parent (the child's behaviour is reinforced), and the parent's behaviour is negatively reinforced by the cessation of the shouting. ADHD children are more demanding of parental time and attention. In small ways like this. the difficult behaviours of ADHD children become reinforced and develop into problems for the family, particularly feelings that the child is "uncontrollable".

This is not helped because ADHD children have altered reinforcement and extinction proceses (ie: longer time to learn new behaviour and remove old behaviour), according to Sagvolden et al 2005).

Parenting of ADHD children can thus be a challenge, but a number of the parents will themselves have ADHD leading to less patience and responsiveness to the child, problems in maintaining attention during supervision, and difficulties in organising tasks, for example (Sagvolden et al 2005).

[6] I am reminded of Hans Eysenck's explanation of extravert personality as due to the motivation to find stimulation because of the lack of arousal in the brain.

PARENTS' EXPERIENCE

Prentice (1996) outlined how both the child themselves and their parents can experience a downward spiral of self-esteem through ADHD. The child struggles to sustain attention at school which leads to poor marks, and, in turn, negative labelling by teachers and teasing by peers. These produce frustration and aggression, and reinforce the problems while self-esteem falls. For the parents, the decline in self-esteem relates to the apparent inability to control their ADHD child. This produces a stricter regime which does not work, and they blame themselves for failing as parents.

Harborne et al (2004) interviewed mothers of nine boys with ADHD (8-11 years old) and one father. Three themes emerged:

- Blame for child's behaviour (eg: poor parenting);
- Battles with professionals, partners and family over diagnosis and treatment;
- Emotional distress.

Many children are diagnosed with ADHD after school age. This can be because the symptoms are noticed for the first time in the classroom environment, or because the parents do not recognise them at home. Maniadaki et al (2006) found that parents with pre-school children who showed ADHD behaviours tended to perceive such behaviours as part of normal development. The researchers investigated just under 600 parents in Athens, Greece with children at nursery schools. The parents filled out questionnaires about their child's behaviour, and responded to vignettes like, "John is a restless five-year old boy who wants to move all the time even when he makes a puzzle or he takes his dinner".

The finding that parents do not see their child's "ADHD" behaviour as a problem is contrary to other research where parents perceived such behaviour as a serious problem. But much of the latter research is with parents of school-aged children.

COGNITIVE DIFFERENCES, AND EDUCATIONAL PERFORMANCE

There is debate over the aspects of attention that are impaired in ADHD. There is the "basic" attentional processes that focus on certain stimuli and not others (selective attention), and the "higher order" processes of control of attention ad sustained attention.

In the laboratory, selective attention is tested by searching for targets in an array (eg: letter "T" mixed with other letters). Sustained attention is measured by tests that involve concentration for long periods, and control of attention involves tasks that change their rules. For example, when a red background screen appears on the computer, choose the left option of two. Sustained attention requires the child to do this continually, but control of attention tasks add another layer like when blue background screen appears choose right option of two. A commonly used set of task is the Test of Everyday Attention in Children (TEA-Ch) (Manly et al 2001) (table 7).

Manly et al (2001) found that ADHD children were poorer than controls on three sustained attention tasks and one control of attention task, but not the selective attention task. However these findings and TEA-Ch have been challenged (Wilding 2005).

TYPE OF ATTENTION	EXAMPLE OF TASK
Selective (2 tasks)	• "Sky Search" - time to find target • "Map Mission" - number of targets found in specific time
Sustained (5 tasks)	• Walk/Don't Walk" - tests ability to continue responding during prolonged concentration
Control (2 tasks)	• "Creature Counting" • "Opposite Worlds" - both test ability to switch to another response after period of one response

Table 7 - Example of tasks in TEA-Ch.

Research has suggested that children with ADHD are poorer at recognising facial expressions of emotion. Yuill and Lyon (2007) compared nineteen 5-11 year-old boys with ADHD from a specialist ADHD clinic in south-east England with nineteen age-matched typically-developing boys from the same geographical area. The children were presented with six pictures of "Thomas" (11 year-old boy) and asked to choose the correct picture associated with an emotion (eg: finding mouldy yoghurt in his lunch-box - disgust). The control task involved six more pictures and non-emotional events (eg: sticking plaster on face as evidence of scratch). Both types of task involved inferences.

The boys with ADHD did poorer than the controls on both tasks, but more so on the emotional task. But why did the ADHD boys score less than the controls? Was it due to impulsivity in answering or an underlying

cognitive deficit?

In the second experiment, Yuill and Lyon (2007) included an "inhibitory scaffolding" procedure to stop impulsive answers. In the same design as above, the inhibitory scaffolding procedure involved the experimenter pointing to each picture before the child could answer the question. Seventeen more boys (5-11 years old) with ADHD were used, but the control group was younger (5-6 years old). The ADHD group did as well as the controls on the non-emotional task, but still poorer on the emotional task. Though they scored higher than the ADHD group in the first experiment (table 8). The inhibitory scaffolding aided their performance.

The researchers felt that the ADHD children had problems drawing inferences about emotions and matching them to emotional expressions.

	NON-EMOTIONAL TASK	EMOTIONAL TASK
Study 1: ADHD Control	 3.16 5.95	 1.79 5.31
Study 2: ADHD Control	 5.65 5.07	 2.53 4.31

(After Yuill and Lyon 2007).

Table 8 - Mean scores (out of six) on two types of task.

In an extensive study, Rodriguez et al (2007) analysed data from Sweden, Denmark and Finland (Nordic Network on ADHD) on over 13 000 first-born 7-8 and 10-12 year-olds and their school performance. Teachers rated inattention and hyperactivity. These core ADHD symptoms were associated with impairments in reading, writing and mathematics. For example, children rated as showing inattention were over eight times more likely to have writing impairments and over seven times more likely to have reading and mathematics problems than children not rated inattentive (table 9).

IMPAIRMENT: *	READING	WRITING	MATHEMATICS
INATTENTION	7.5	8.2	7.3
HYPERACTIVE	3.4	3.7	3.4

(My calculation of mean of three countries for odds ratios adjusted for maternal education, family structure, and gender)

Table 9 - Odds ratios of impairments based on symptoms.

ADULT PROBLEMS

"Full" ADHD exists in 4% of the adult population, while 20-50% of child sufferers show "residual symptoms" in adulthood (Klein and Mannuazza 1991). Faraone et al (2006) performed a meta-analysis on thirty-two studies, and found that ADHD at age 25 depends on the definition used. When "persistent ADHD" used, around 15% of childhood ADHD sufferers are still suffering from it, but the figure is nearer two-thirds when "ADHD in partial remission" is used.

Some symptoms decline more than others by the late teens - eg: hyperactivity by 50% but inattention only by 20% (Wiles et al 2002).

Many children diagnosed with ADHD have other psychiatric problems as adolescents and adults (Sagvolden et al 2005). Biederman et al (2006) compared 112 6-18 year-old children with ADHD (DSM-IIIR criteria) ten years later with 105 controls for any mental disorder. The former were six times more likely to suffer any major psychopathology.

Childhood ADHD has been linked to adult problem and criminal behaviour in a number of studies. For example, among seventy-five male inmates at a Swedish young offenders institute, who had committed a total of 12 000 crimes, 68% had ADHD, and those who showed hyperactivity at the youngest age (before seven years old) had committed more crimes on average (Dalteg and Lavander 1998).

While boys with ADHD were more likely to develop anti-social personality disorder and substance abuse in adulthood than controls (Mannuzza et al 1998).

But what is the relationship between childhood ADHD and adult behaviour? One suggestion is that ADHD and anti-social behaviours are two points of the same process (eg: Patterson et al 2000). Or ADHD is a predictor of adult personality disorders (eg: Fossati et al 2002).

The issue can be that adults who show problem behaviours were not diagnosed as children with ADHD, and so diagnosis is then done in retrospect (with, for example, the Wender Utah Rating Scale; WURS; Ward et al 1993; table 10).

Individuals respond to 25 items on a five-point scale (0 = "not at all or very slightly" to 4 = "very much") using the sentence stem: "As a child I was or had.."

- Acting without thinking, impulsive
- Tend to be immature
- Feel guilty, regretful
- Lose control of myself
- Tend to be or act irrational
- Unpopular with other children, didn't keep friends for long, didn't get along with other children

(Source: McCann et al 2000)

Table 10 - Wender Utah Rating Scale.

Giotakis et al (2005) used the WURS (among other questionnaires) with forty-four male convicted rapists in Greece. Twenty-seven rapists who scored 46 or more on the WURS (cut-off point for childhood ADHD diagnosis) showed more aggression (self and other reported), more impulsivity, greater novelty-seeking behaviour, and less co-operation than the other rapists in the study (table 11).

	WURS SCORE >46 (CHILDHOOD ADHD)	WURS SCORE <46 (NO ADHD)
Aggression * &	106	88
Impulsivity ** &&	90	72
Co-operation *** &&&	49	57
Novelty-seeking **** &&	74	60

(* Higher score = more aggression; ** higher score = more impulsive; *** higher score = more co-operative; *** higher score = more novelty-seeking behaviour; & p<0.001; && p<0.0001; &&& p<0.02)

(After Giotakis et al 2005)

Table 11 - Mean scores, on behaviours showing significant differences, of rapists diagnosed with childhood ADHD or not.

CRITICAL VOICES

Baughman (2003) described ADHD as an "epidemic" with diagnoses rising in the USA from 150 000 children in 1970 to six million in 2002.

Scott (2000) saw the need for a quick solution as involved in the increase in diagnosis and medication: "if a child is behaving so badly at school that he is threatened with exclusion.. there will be considerable

pressure to improve behaviour by the quickest means available. Stimulants act more quickly than psychotherapy" (p45).

There was concern about the increase in diagnosis of ADHD and prescription of stimulant medication in Australia, particularly Western Australia, in the early part of the century. Berbatis et al (2002), using official statistics, reported that 4.2-4.5% of 4-17 year-olds in Western Australia received stimulant medication ("psychostimulants") in 2000 [7].

In this context, McHoul and Rapley (2005) used discourse analysis with a consultation in Western Australia between a paediatrician, parents and their son ("Alan") over the diagnosis of school problems and treatment. The authors were concerned that the "possibility" of the ADHD was enough for medication (ie: diagnosis is not an objective process of true or false, but is highly subjective).

The doctor preferred the diagnosis of ADHD during the consultation while the parents resisted this diagnosis. This led the doctor to suggest a "trial of medication and we'll see what happens..". McHoul and Rapley (2005) noted: "Now this is peculiar in some respects: the doctor is still not certain but wants to see if the response to drugs brings about a concomitant improvement in behaviour. It's almost as if Alan is to become a guinea pig: maybe he's ADHD, maybe not. But if so, the administration of drugs will tell us" (pp433-434).

By the end of the two-hour consultation, the doctor had "won" the argument through the use of "evidence" (though this was contested at length by the parents).

This study shows how behaviour is socially constructed, and specifically how a diagnosis of ADHD is created through interaction and talk. The social construction (or, more specifically, discursive psychology) position challenges the existence of objective truths (like a real disorder) and emphasises that behaviour takes place within a social context that dictates its meaning and understanding. Thus ADHD is a social construct in the same way as all other labels on behaviour as normal/abnormal, good/bad, desirable/undesirable, and so on.

Following the tradition of ideas in sociology like symbolic interactionism, the issues is not whether the child has ADHD or not, but more like, what is the purpose

[7] Berbatis et al (2002) believed that there was a link between the increase in licit amphetamine prescriptions in this medication, and the increase in illicit amphetamine use (eg: "speed") in Australia. However, Witens et al (2002) reported that medication reduced by over half the risk of co-morbidity of substance abuse in later adolescence in a five-year longitudinal study.

for society of the use of concepts like ADHD in making sense of children's behaviour?

When behaviour once viewed as non-medical is redefined as medical, non-medical individuals become involved in "disseminating understanding of the new sickness" (Phillips 2006). In the case of ADHD, teachers have been "recruited" to diagnose the condition and recommend treatment. This is not an official recruitment by an organisation, but the simple fact that teachers see children for long periods and become aware of their behaviour. The teacher has become "the sickness and treatment broker for ADHD" (Phillips 2006). This can include the actual administration of medication to the child during the school day.

Phillips (2006) is further concerned by the role of pharmaceutical companies in influencing teachers. Collaborative projects on ADHD awareness like "Novartis" (manufacturers of "Ritalin") with the National Association of School Nurses in the USA in 1997 "reinforce the place of the pharmaceutical industry as a benevolent and authoritative presence within the school" (Phillips 2006). This issue is as important, for her, as the ethics of fast-food marketing within schools.

TREATMENT

One of the most used treatments is medication with stimulants, which, as mentioned earlier is controversial. The logic of giving stimulants to hyperactive and impulsive children is that the frontal cortex is underactive (the brake on impulsive behaviour). Amphetamine-based stimulants increase activity in the area of the brain, and strengthen the brake on hyperactive and impulsive behaviour.

The key question with any treatment is whether it works. The MTA study in the USA followed 579 children (aged 7-10 years old) with DSM-IV combined sub-type ADHD over fourteen months (Jensen et al 1999).

The children were randomised to (i) medication only, (ii) intensive behavioural treatment, (iii) combination of both, and (iv) "standard community care" (control group). Nineteen measures of the children's behaviour were collected. All groups showed improvements between the beginning and the end of the study with the largest improvements in groups (iii) and (i). The combined treatment group were superior on reduction of aggressive symptoms, teacher-rated social skills, parent-child relations, and reading symptoms. Behavioural treatment was best for improving social skills. It was concluded that, overall, medication was best.

Rubia and Smith (2001) noted some of the criticisms of the study:

a) Design problems - eg: behavioural treatment for 4-5 months only.

b) Parents in all groups used some form of behavioural treatment.

c) In the combined group, medication was given before the behavioural treatment.

d) The level of medication was 20% less in the combined than medication only group.

e) Sample included "only children".

In terms of which medication is better, Faraone et al (2002) performed a meta-analysis on four studies comparing "Adderall" (amphetamine) and methylphenidate (eg: "Ritalin"). "Adderall" was more effective on all measures of ADHD symptoms, and by who did the rating (ie: parent, teacher, clinician).

Behavioural treatments using operant conditioning are the main alternative to medication.

Weeks and Laver-Bradbury (1997) reported the use of a behaviour modification programme in Hampshire with three year-olds. It included teaching parents to keep calm, give clear messages and boundaries, and use distraction techniques for temper tantrums. Parents rated the child's problem before the programme and after eight weeks. All problem behaviours were reduced; eg: disobedience by 39%, overactivity by 22%. But the control group also showed improvements (eg: disobedience reduced by 28% and overactivity by 24%). This may have been due to supportive discussions with the researchers which motivated the parents.

Chu (2003b) described multi-faceted interventions that combine different components:

- Education and training of parents to recognise symptoms and how to deal with them;
- Environmental adaptation - changing environments the child (eg: calming colour scheme);
- Behavioural treatments to reduce impulsivity and hyperactivity;
- Medication;
- Social skills training and other compensatory strategies.

REFERENCES

Achenbach, T.M (1991) Child Behaviour Checklist Burlington, VT: University of Vermont, Dept of Psychiatry

APA (1968) Diagnostic and Statistical Manual of Mental Disorders (2nd ed) DSM-II Washington DC: American Psychiatric Association

APA (1980) Diagnostic and Statistical Manual of Mental Disorders (3rd ed) DSM-III Washington DC: American Psychiatric Association

APA (1987) Diagnostic and Statistical Manual of Mental Disorders (3rd ed revised) DSM-IIIR Washington DC: American Psychiatric Association

APA (1994) Diagnostic and Statistical Manual of Mental Disorders (4th ed) DSM-IV Washington DC: American Psychiatric Association

Barkley, R.A (1990) ADHD: A Handbook for Diagnosis and Treatment New York: Guilford

Barkley, R.A (1998) A theory of ADHD: Inhibition, executive functions, self-control and time. In Barkley, R.A (ed) ADHD: A Handbook for Diagnosis and Treatment (2nd ed) New York: Guilford

Barkley, R.A et al (2002) International consensus statement on ADHD Clinical Child and Family Psychology Review 5, 89-111

Baughman, F.A (2003) The rise and fall of ADD/ADHD Journal of Critical Psychology, Counselling and Psychotherapy Summer, 89-95

Berbatis, C.G et al (2002) Licit psychostimulant consumption in Australia, 1984-2000: International and jurisdictional comparison Medical Journal of Australia 177, 539-543

Biederman, J et al (2006) Young adult outcome of attention deficit hyperactivity disorder: A controlled ten-year follow-up study Psychological Medicine 36, 167-179

Brookes, K.J et al (2006) The analysis of 51 genes in DSM-IV combined type attention deficit hyperactivity disorder: Association signals in DRD4, DAT1 and 16 other genes Molecular Psychiatry 11, 934-953

Cannon, M et al (2004) ADHD is best understood as a cultural construct: Introduction British Journal of Psychiatry 184, p8

Chess, S (1960) Diagnosis and treatment of the hyperactive child New York State Journal of Medicine 60, 2379-2385

Chu, S (2003a) Attention deficit hyperactivity disorder (ADHD) part one: A review of the literature International Journal of Therapy and Rehabilitation 10, 5, 218-227

Chu, S (2003b) Attention deficit hyperactivity disorder (ADHD) part two: Evaluation and intervention International Journal of Therapy and Rehabilitation 10, 6, 254-263

Clements, S & Peters, J (1962) Minimal brain dysfunction in the school-age child Archives of General Psychiatry 6, 185-197

Dalteg, A & Lavander, S (1998) 12 000 crimes by 75 boys Journal of Forensic Psychiatry 9, 1, 39-57

DuPaul, G.J et al (1998) ADHD Rating Scale New York: Guilford

Faraone, S.V et al (2002) Comparative efficacy of adderall and methylphenidate in Attention-deficit/Hyperactivity Disorder: A meta-analysis Journal of Clinical Psychopharmacology 22, 5, 468-473

Faraone, S.V et al (2006) The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies

Psychological Medicine 36, 159-165

Feingold, B (1975) Why Your Child is Hyperactive New York: Random House

Fossati, A et al (2002) History of childhood ADHD symptoms and borderline personality disorder: A controlled study Comprehensive Psychiatry 43, 369-377

Giotakis, O et al (2005) Aggression, impulsivity, and plasma sex hormone levels in a group of rapists, in relation to their history of childhood attention-deficit/hyperactivity disorder symptoms Journal of Forensic Psychiatry and Psychology 16, 2, 423-433

Harborne, A et al (2004) making sense of ADHD: A battle for understanding? Parents' views of their child being diagnosed with ADHD Clinical Child Psychology and Psychiatry 9, 3, 327-339

Jensen, P.S et al (1999) A fourteen-month randomised clinical trial of treatment strategies for attention-deficit/hyperactivity disorder Archives of General Psychiatry 56, 1073-1086

Jensen, P.S et al (2001) Findings from the NIMH Multi-Modal Treatment Study (MTA): Implications and applications for primary care providers Journal of Developmental and Behavioural Paediatrics 22, 60-73

Kahn, E & Cohen, L (1934) Organic drivenness: A brain stem syndrome and an experience with case reports New England Journal of Medicine 210, 748-756

Keith, R.W (1994) Auditory Continuous Performance Test San Antonio, TX: Psychological Corporation

Klein, R & Mannuzza, S (1991) Long-term outcome of hyperactive children: A review Journal of the American Academy of Child and Adolescent Psychiatry 30, 383-387

Laufer, M et al (1957) Hyperkinetic Impulse Disorder in children's behaviour problems Psychosomatic Medicine 19, 38-49

Maniadaki, K et al (2006) Parental beliefs about the nature of ADHD behaviours and their relationship to referral intentions in preschool children Child Care, Health and Development 33, 2, 188-195

Manly, T et al (2001) The differential assessment of children's attention: The Test of Everyday Attention in Children (TEA-Ch), normative sample and ADHD performance Journal of Child Psychology and Psychiatry 42, 1065-1081

Mannuzza, S et al (1998) Adult psychiatric status of hyperactive boys grown up American Journal of Psychiatry 155, 493-498

Mayes, R & Rafalovich, A (2007) Suffer the restless children: The evolution of ADHD and paediatric stimulant use, 1900-80 History of Psychiatry 18, 4, 435-457

McCann, B.S et al (2000) Discriminant validity of Wender Utah Rating Scale for attention-deficit/hyperactivity disorder in adults Journal of Neuropsychiatry and Clinical Neurosciences 12, 240-245

McHoul, A & Rapley, M (2005) A case of attention-deficit/hyperactivity disorder diagnosis: Sir Karl and Francis B. slug it out on the consulting room floor Discourse and Society 16, 3, 419-449

Palmer, E.D & Finger, S (2001) An early description of ADHD (inattentive subtype): Dr.Alexander Crichton and "mental restlessness" Child Psychology and Psychiatry Review 6, 2, 66-73

Patterson, G.R (2002) The early development of coercive family process. A developmental analysis and model for intervention. In Reid, J.B et al (eds) Anti-Social Behaviour in Children and Adolescents Washington

DC: American Psychological Association

Patterson, G.R et al (2000) Hyperactive and anti-social behaviours: Co-morbidity or two points in the same process? Developmental Psychopathology 12, 91-106

Phillips, C.B (2006) Medicine goes to school: Teachers as sickness brokers for ADHD PLoS Medicine 3, 4, e182

Prentice, P (1996) Attention deficit hyperactivity disorder Psychology Review November, 20-24

Rafalovich, A (2005) Exploring clinician uncertainty in the diagnosis and treatment of ADHD Sociology of Health and Illness 27, 3, 305-323

Rodriguez, A et al (2007) Do inattention and hyperactivity symptoms equal scholastic impairment? Evidence from three European cohorts BMC Public Health 7, 327

Rubia, K & Smith, A (2001) Attention deficit-hyperactivity disorder: Current findings and treatment Current Opinion in Psychiatry 14, 4, 309-316

Sagvolden, T et al (2005) A dynamic developmental theory of attention-deficit/hyperactivity disorder predominantly hyperactive/impulsive and combined subtypes Behavioural and Brain Sciences 28, 397-419

Schrag, P & Divoky, D (1975) The Myth of the Hyperactive Child and Other Means of Child Control New York: Pantheon

Scott, S (2000) Bad behaviour New Scientist 20/5, 44-45

Singh, I (2008) Beyond polemics: Science and ethics of ADHD Nature Reviews Neuroscience 9, 957-964

Still, G (1902) The Goulstonian Lectures on some abnormal psychical conditions in children Lancet 1, 1008-12 & 1079-82 & 1163-1167

Swanson, J et al (1998) ADHD and hyperkinetic disorder Lancet 351, 429-433

Taylor, E (2004) ADHD is best understood as a cultural construct: Against British Journal of Psychiatry 184, p9

Timimi, S (2004) ADHD is best understood as a cultural construct: For British Journal of Psychiatry 184, 8-9

Tredgold, A (1922) Mental Deficiency (Amentia) (4th ed) New York: William Wood & Co

Voeller, K.S (2001) Attention-deficit/hyperactivity disorder as a frontal-subcortical disorder. In Lichter, D.G & Cummings, J.L (eds) Frontal-Subcortical Circuits in Psychiatric and Neurological Disorders New York: Guilford

Waldman, I.D & Gizer, I.R (2006) The genetics of attention deficit hyperactivity disorder Clinical Psychology Review 26, 4, 396-432

Ward, M.F et al (1993) The Wender Utah Rating Scale: An aid in the retrospective diagnosis of childhood attention deficit hyperactivity disorder American Journal of Psychiatry 150, 885-890

Weeks, A & Laver-Bradbury, C (1997) Behaviour modification in hyperactive children Nursing Times 19/11, 56-58

WHO (1992) International Classification of Diseases (10th ed) ICD-10 Geneva: World Health Organisation

Wilding, J.A (2005) Is attention impaired in ADHD? British Journal of Developmental Psychology 23, 487-505

Wilens, T.E et al (2002) Attention deficit/hyperactivity disorder

across lifespan Annual Review of Medicine 53, 113-131

Yuill, N & Lyon, J (2007) Selective difficulty in recognising facial expressions of emotion in boys with ADHD: General performance impairments or specific problems in social cognition? European Child and Adolescent Psychiatry 16, 6, 398-404

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